4	CAS No.: 110-49-6	Substance: 2-Methoxyethyl acetate

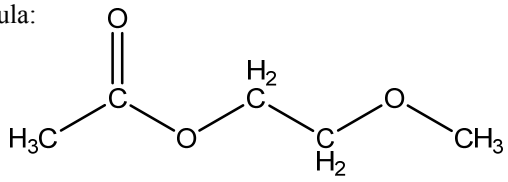
Chemical Substances Control Law Reference No.: 2-740（Ethylene glycol monoalkyl (C=1–4) ether acetate ester)

PRTR Law Cabinet Order No.: 1-135

Molecular Formula: $C_5H_{10}O_3$

Molecular Weight: 118.13

Structural Formula:



1. General information

This substance is freely miscible with water, the partition coefficient (1-octanol/water) (log K_{ow}) is 0.10 (calculated value), and the vapor pressure is 7 mmHg (900 Pa) (20°C). The biodegradability (aerobic degradation) is not thought to be limited. Furthermore, its half-life for hydrolysis is 39 days at pH 8 and 391 days and pH 7.

This substance is designated as a Class 1 Designated Chemical Substance under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (PRTR Law). The main use of this substance is as an electronic material cleaning solvent. It is also used as a solvent for printing inks, coatings, and adhesives used for metal sheets. The production and import quantity of ethylene glycol monoalkyl (C=1–4) ether acetate ester in fiscal 2013 was less than 1,000 t. The production and import category under the PRTR Law is more than 100 t.

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2. Exposure assessment

Total release to the environment in fiscal 2013 under the PRTR Law was approximately 9.7 t, and all releases were reported. The major destination of reported releases was the atmosphere. In addition, approximately 8.6 t was transferred to waste materials. Industry types with large reported releases were metal product manufacturing and electrical machinery manufacturing for the atmosphere and the chemical industry for public water bodies. A multi-media model used to predict the proportions distributed to individual media in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment overall or to the atmosphere in particular, the predicted proportion distributed to the atmosphere was 35.9%, that distributed to water bodies was 33.8%, and that distributed to soil was 30.1%. In regions where the largest quantities were estimated to have been released to public water bodies, the predicted proportion distributed to water bodies was 87.5%.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was less than around 0.02 μg/m^3. In addition, the predicted maximum concentration for indoor air was around 13 μg/m^3, albeit from past data. The mean annual value for the atmospheric concentration in fiscal 2013 was calculated by using a plume-puff model on the basis of releases to the atmosphere reported according to the PRTR Law; this model predicted a maximum level of 0.63 μg/m^3. However, releases less than around 10 times that of the releases reported under the PRTR law were estimated as part of a refinement process carried out for the VOC release inventory.

Information to determine the maximum expected oral exposure could not be obtained. However, past data from public freshwater bodies yielded a maximum expected exposure of less than 0.028 μg/kg/day. When releases to public freshwater bodies in fiscal 2013 reported according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers by taking into consideration only dilution gave a maximum value of 0.76 μg/L. Using this estimated

concentration for rivers to calculate oral exposure gave 0.030 μg/kg/day. The risk of exposure to this substance by intake from an environmental medium via food is considered slight, given the low bioaccumulation of the substance expected on basis of its physicochemical properties.

Information to determine the predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not be obtained. However, past data yielded values of less than 0.7 μg/L for public freshwater bodies and less than around 0.7 μg/L for seawater. When releases to public freshwater bodies in fiscal 2013 reported according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers by taking into consideration only dilution gave a maximum value of 0.76 μg/L.

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3. Initial assessment of health risk

This substance may cause effects on the bone marrow and central nerve system. At high levels, the substance may cause effects on the blood, which may result in lesions of blood cells and kidney impairment. Inhalation exposure to the substance causes dizziness, drowsiness and headache. Oral exposure causes abdominal pain, nausea, vomiting, weakness and unconsciousness in addition to the symptoms observed when inhaled.

The substance is mildly irritating to the eyes, and contact with the eyes causes redness. Contact with the skin causes dryness and the substance may be absorbed to cause the same symptoms as when inhaled.

As sufficient information on the carcinogenicity of the substance was not available, the initial assessment was conducted on the basis of information on its non-carcinogenic effects.

The NOAEL of 250 mg/kg/day for oral exposure (based on testis weight loss, lower sperm count, etc.), determined from reproductive and developmental toxicity tests in mice, was adjusted for exposure conditions to obtain 179 mg/kg/day, and subsequently divided by a factor of 10 to account for extrapolation from sub-acute to chronic exposure. The obtained value of 18 mg/kg/day was deemed to be the lowest reliable dose and was identified as the ‘non - toxic level*’ of the substance for oral exposure.

The ‘non-toxic level*’ for inhalation exposure could not be identified.

With regard to oral exposure, owing to lack of identified exposure levels, the health risk could not be assessed. The oral exposure level of the substance was calculated to be less than 0.028 μg/kg/day, using the maximum concentration in public freshwater bodies reported in 1986. The MOE (Margin of Exposure) would be over 64,000, when calculated from this exposure level and the ‘non-toxic level*’of 18 mg/kg/day, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans.

In addition, the maximum exposure level was calculated to be 0.030 μg/kg/day. This value derives from the concentrations of the substance in the effluents from high discharging plants, estimated according to the emissions data reported in FY 2013 under the PRTR Law. The MOE derived from this value and the ‘non-toxic level*’ was 60,000. Since exposure to the substance in environmental media via food is presumed to be limited, its inclusion in the calculation would not change the MOE significantly. Therefore, collection of further information would not be required to assess the health risk of this substance via oral exposure.

With regard to inhalation exposure, owing to lack of identified ‘non-toxic level*’, the health risk could not be assessed. Assuming 100% of the ingested substance is absorbed, the ‘non-toxic level*’ of inhalation exposure, derived by converting that of oral exposure, would be 60 mg/m^3. The MOE would be over 300,000, when calculated from the converted ‘non-toxic level*’ of inhalation exposure and the predicted maximum exposure concentration of less than 0.02 $\mu g/m^3$ in ambient air, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans.

In addition, the maximum concentration (annual mean) in ambient air near the operators releasing large amount of the substance was estimated to be 0.63 $\mu g/m^3$ on the basis of the data reported in FY 2013 under the PRTR Law. The MOE would be 9,500, when calculated from the maximum concentration in ambient air and the

'non-toxic level*'. The MOE would still be over 100, even when using the estimated emission level of the substance according to the consideration for the elaboration of the VOC emission inventory (slightly less than 10 times as much as the emission level reported under the PRTR Law). On the other hand, the MOE would be 460, when calculated from the maximum exposure concentration in indoor air of 13 μg/m^3 reported in 2001. Therefore collection of further information would not be required to assess the health risk of this substance via inhalation both in ambient and indoor air.

Toxicity				Exposure assessment		Result of risk assessment			Judgment
Exposure Path	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration				
Oral	'Non-toxic level*' 18 mg/kg/day	Mouse	Testis weight loss, lower sperm count etc.	Drinking water	— μg/kg/day	MOE	—	×	(○)
				Groundwater	— μg/kg/day	MOE	—	×	
Inhalation	'Non-toxic level*' — mg/m^3	—	—	Ambient air	<0.02 μg/m^3	MOE	—	×	(○)
				Indoor air	— μg/m^3	MOE	—	×	(○)

Non-toxic level *

- When a LOAEL is available, it is divided by 10 to obtain a NOAEL-equivalent level.
- When an adverse effect level for the short-term exposure is available, it is divided by 10 to obtain a level equivalent to an adverse effect level for the long-term exposure.

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4. Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC_{50} of 7,900,000 μg/L for growth inhibition in the green algae *Pseudokirchnerella subcapitata*, a 48-h EC_{50} of 245,400 μg/L for swimming inhibition in the crustacean *Daphnia magna*, and a 96-h LC_{50} of 40,000 μg/L in the fish species *Menidia beryllina* (inland silverside). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 400 μg/L was obtained.

With regard to chronic toxicity, the following reliable data was obtained: a 72-h NOEC of 3,100,000 μg/L for growth inhibition in the green algae *P. subcapitata.* Accordingly, based on this chronic toxicity value and an assessment factor of 100, a PNEC of 31,000 μg/L was obtained.

The value of 400 μg/L, obtained from the acute toxicity to the fish species, was used as the PNEC for this substance.

Information to determine the PEC of this substance could not be obtained. As such, a judgment on ecological risk could not be made. However, past data yielded a value of less than around 0.7 μg/L for both freshwater bodies and seawater. The ratio of each PEC value to the PNEC is less than 0.01. When releases to public freshwater bodies in fiscal 2013 reported according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers by taking into consideration only dilution gave a maximum value of 0.76 μg/L. The ratio of this value to the PNEC is also less than 0.01. Accordingly, there is little need to collect new data regarding this substance.

Hazard Assessment (Basis for PNEC)			Assessment Coefficient	Predicted no effect concentration PNEC (μg/L)	Exposure Assessment		PEC/PNEC ratio	Judgment based on PEC/PNEC ratio	Assessment result
Species	Acute/ chronic	Endpoint			Water body	Predicted environmental concentration PEC (μg/L)			
Fish (inland silverside)	Acute	LC_{50} mortality	100	400	Freshwater	—	—	×	○
					Seawater	—	—		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Although risk to human health could not be confirmed, collection of further information would not be required.	(○)
	Inhalation exposure	Although risk to human health could not be confirmed, collection of further information would not be required.	(○)
Ecological risk	No need for further work at present.		○

［Risk judgments］ ○: No need for further work ▲: Requiring information collection

■: Candidates for further work ×: Impossibility of risk characterization

（○）: Although risk to human health could not be confirmed, collection of further information would not be required.

（▲）: Further information collection would be required for risk characterization.